CASE REPORTS

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Runaway Pacemaker

A Serious and Unresolved Problem

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THE TREATMENT of certain cardiac arrhythmias with permanent intracardiac pacemakers is a widely used therapeutic modality. While technologic advances have increased the safety and effectiveness of these devices, serious complications still occur.[1-3] The purpose of this communication is to emphasize the continued importance and seriousness of pacemaker-induced ventricular tachycardia (runaway pacemaker).[4-7]

Reports of Cases

CASE 1. A 76-year-old man was brought to the emergency room at San Francisco General Hospital on January 17, 1973 in an unresponsive state. Four months before admission he had been evaluated at another hospital for prostatic carcinoma. During that admission, results of cardiac evaluation were unremarkable except for electrocardiographic findings, which showed sinus rhythm, a P-R interval of 0.20 seconds and right bundle branch block. One month before admission, the patient experienced postural lightheadness and dyspnea on exertion and on electrocardiogram showed complete A-V block with a ventricular response of 32 beats per minute. A permanent transvenous Cordis unipolar demand pacemaker (ECTOCOR catalogue No. 304-070, model 114c7) was implanted. There were no complications, and the patient's physician, who saw him weekly, noted a regular cardiac rhythm at a rate of 72 beats per minute.

On the morning of admission the patient was found unresponsive in the street, and resuscitative measures were initiated on route to the hospital. On physical examination the pulse and blood pressure were unobtainable, and the pupils were dilated and unresponsive. The initial rhythm electrocardiographic strip showed a pacemaker discharge rate of 270 beats per minute without ventricular response (Figure 1A). A chest incision was made and the pacing wire was disconnected from the implanted generator; the electrocardiographic tracing then showed a slow idioventricular rhythm (Figure 1B). A temporary external generator was connected to the existing electrode and electrical

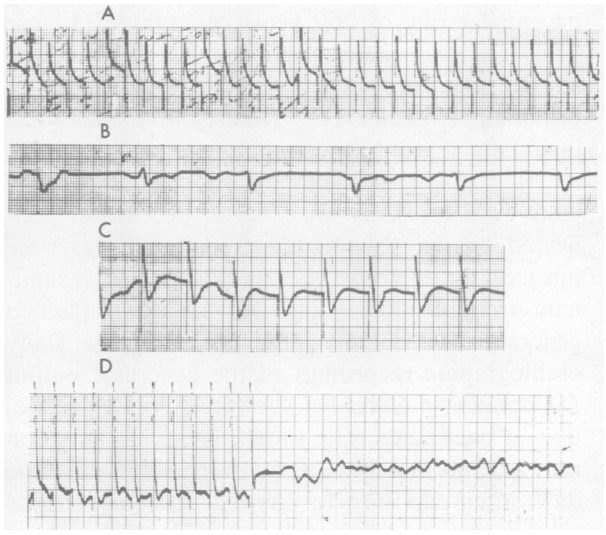

Figure 1.—(A-C) Electrocardiographic rhythm strips obtained in Case 1 show **(A)** a runaway pacemaker at a rate of 270 beats per minute, **(B)** idioventricular rhythm at a rate of approximately 40 beats per minute after disabling the malfunctioning pacemaker and **(C)** while pacing with an external pacemaker connected to the intracardiac electrode catheter. An electrocardiographic rhythm strip **(D)** obtained in Case 2 shows a runaway pacemaker with a discharge rate of 260 beats per minute; ventricular fibrillation occurred after the implanted pacemaker was disconnected.

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capture was accomplished (Figure 1C), but vital signs never returned.

On postmortem examination, left ventricular hypertrophy, extensive myocardial fibrosis and severe diffuse atherosclerotic coronary artery disease were seen. The pacemaker electrode tip was in place behind the anterior papillary muscle of the right ventricle with "no unusual fibrosis or tissue reaction." The generator was removed, exposed to room air for 72 hours, and connected to a Tektronix® oscilloscope and interval counter. The output of the generator was 6.5 millamperes (ma) when connected to a 500 ohm resistor. The pulse generator produced an impulse of normal contour and of 1.5 millisecond (msec) duration at a rate of 70 beats per minute.

CASE 2. A 73-year-old man was brought to the emergency room at San Francisco General Hospital on June 12, 1974 in an unresponsive state with no vital signs. Six months before admission, a Cordis transvenous unipolar demand intracardiac pacemaker (Stanicor No. 143 E7-16663) was implanted at a rate of 72 beats per minute because of chronic congestive heart failure and complete A-V block. No complications occurred until the day of admission when, while shopping, the patient collapsed.

On admission, an electrocardiogram showed a pacemaker rate of 260 beats per minute (Figure 1D). The pacemaker generator was disconnected and an electrocardiogram showed ventricular fibrillation (Figure 1D). Resuscitative measures, including direct current cardioversion and attempts to pace the heart during periods of complete A-V block by an external pacemaker connected to the existing electrode catheter, were unsuccessful. Permission for postmortem examination could not be obtained, but the implanted generator was removed for further study. Daily oscillographic recordings of the generator output (as detailed previously) showed a gradual reduction in pacemaker rate until control implantation rate and normal wave form were achieved three days after exposure to air. The pacemaker was submerged in saline solution for 48 hours at 37°C (98.6°F); no change in form or rate of the generator impulse occurred.

Discussion

Pacemaker-induced ventricular tachycardia usually results from primary electronic component failure: defective capacitors, transistors, or transformers as well as leakage of biologic fluid into the generator box, which all may produce malfunction of the oscillator circuit, are believed to be important causes of component failure leading to a "runaway pacemaker."[8] In the cases reported here, the resumption of normal pacemaker function after exposure of the generators to air supports the concept of component failure (probably related to influx of tissue fluids) rather than primary generator malfunction. Immersion of the generator, extracted from the second patient, in saline solution for 48 hours failed, however, to produce an increased rate of pacemaker discharge.

Both pacemakers were returned to the manufacturer, but the precise cause of the malfunction still remains uncertain. In 1970, Wallace and coworkers[7] reviewed the 44 recorded instances of runaway pacemaker and found that this complication developed in pacemakers made by all of the major producers (and at all pacing modes) and was associated with a 34 percent mortality rate. Further improvements of pacemaker design have probably resulted in a decreased incidence of this complication; our search of the literature, for example, showed only one additional report of runaway pacemaker since 1970.[3] The present report is of importance because it emphasizes that the problem of pacemaker-induced ventricular tachycardia has not been resolved completely.

The runaway pacemaker complication poses important clinical problems because this complication may occur at any time (two days to 36 months)[9,10] after implantation and may occur without antecedent change in pacemaker discharge rate or sensing function, or change in the generator impulse wave form. The increased rate is associated with diminished generator output, which may or may not result in ventricular capture. In either event, emergency treatment dictates either prompt disabling of the generator and control of the heart rate with preferably an external pacemaker connected to the original intracardiac electrode or infusion of isoproterenol.

Summary

Two patients with implanted permanent transvenous pacemakers had cardiac arrests associated with very rapid pacemaker discharge rates (runaway pacemaker). This complication developed without other evidence of pacemaker failure four months after insertion in one patient and six months after insertion in the other. Both patients died. Exposure of the batteries to room air after 48 hours resulted in return of normal pacing

function. This report documents the persistence and seriousness of this condition and emphasizes the need for prompt recognition and treatment of this emergency.

ADDENDUM: Both batteries were returned to the manufacturer but we have not as yet received the technical analyses of the failed units. Since submission of this manuscript, one additional report of runaway pacemaker associated with a fatal outcome has been published.[11]

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Refer to: Vieweg WVR, Tretheway DG: Transmural myocardial infarction in a young man with normal coronary arteriograms. West J Med 123:53-55, Jul 1975

Transmural Myocardial Infarction in a Young Man with Normal Coronary Arteriograms

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WOMEN SEEM particularly predisposed to angina pectoris and subendocardial infarction without demonstrable coronary artery disease.[1,2] Rarely has a transmural infarction occurred in a young man without other cardiac disease or apparent cause as is described in this report.

Report of a Case

A 29-year-old Navy man was admitted to the hospital because of crushing retrosternal pain associated with shortness of breath and diaphoresis. There was no history of prior chest pain, high blood pressure or lipid disturbance, and no family history of coronary artery disease. Findings on the initial electrocardiogram showed an acute inferolateral transmural myocardial infarction (Figure 1a). Daily serum glutamic oxaloacetic transaminase determinations were 29, 320, 118, 55 and 66 milliunits (mU) per millimeter with a normal range of 7 to 40. The patient's course was uncomplicated.

Two months after infarction, an electrocardio-

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